

Cognitive Disparities: The Impact of the Great Depression and Cumulative Inequality on Later-Life Cognitive Function

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Abstract Population aging has driven a spate of recent research on later-life cognitive function. Greater longevity increases the lifetime risk of memory diseases that compromise the cognitive abilities vital to well-being. Alzheimer’s disease, thought to be the most common underlying pathology for elders’ cognitive dysfunction (Willis and Hakim 2013), is already the sixth leading cause of death in the United States (Alzheimer’s Association 2016). Understanding social determinants of pathological cognitive decline is key to crafting interventions, but evidence is inconclusive for how social factors interact over the life course to affect cognitive function. I study whether early-life exposure to the Great Depression is directly associated with later-life cognitive function, influences risky behaviors over the life course, and/or accumulates with other life-course disadvantages. Using growth curve models to analyze the Health and Retirement Study, I find that early-life exposure to the Great Depression is associated with fluid cognition, controlling for intervening factors—evidence for a critical period model. I find little support for a social trajectory model. Disadvantage accumulates over the life course to predict worse cognitive function, providing strong evidence for a cumulative inequality model.

Keywords Cognitive function · Early-life conditions · Critical periods · Cumulative inequality · Alzheimer’s disease

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Introduction

Cognitive function is a clear predictor of mental health and well-being for elders, indicative of autonomy, identity, and social connectedness (Baltes and Smith 2003). As age structures shift to older populations, age-related cognitive decline will exact an increasing toll on societies (United Nations 2015). For many, cognitive decline will prevent independent living because dementia is a primary determinant of elders' care needs and institutionalization (Deary et al. 2009). The social and economic costs of dementia are high—already \$159 to \$215 billion per year in the United States (Hurd et al. 2013). In light of these dire forecasts and the failure of medical interventions, researchers have focused on social predictors of late-life cognitive function. Dementia appears to come at “the tail end of a long, accumulating pathologic process,” ranging from low-levels of educational attainment to lack of adulthood cognitive stimulation (Liu et al. 2010:493). However, that process and the factors that influence it are not yet well understood.

Much research has focused on specific risk factors for Alzheimer's disease because it has been considered the primary underlying cause of pathological later-life cognitive decline (Willis and Hakim 2013), contributing to 60 % to 80 % of dementias¹ (Alzheimer's Association 2015). Evidence suggests an association between Alzheimer's disease and education (Mayeux and Stern 2012; Meng and D'Arcy 2012; Stern 2012); exercise (Farina et al. 2014); social activity (Reed et al. 2011); and physical health, including stroke (Willis and Hakim 2013), hypertension (Borenstein et al. 2006), body mass index (BMI), and diabetes (Profenno et al. 2010). There is mixed evidence for whether mid- to later-life cognitive function is associated with socioeconomic status (SES) (Cagney and Lauderdale 2002; Kaplan et al. 2001; Luo and Waite 2005; Mocerri et al. 2000; Singh-Manoux et al. 2005, 2011; Turrell et al. 2002; Zeki Al-Hazzouri et al. 2011).

Conclusive evidence for social predictors of later-life cognitive function is lacking partially because many studies control only for age and sex, but also because of significant methodological challenges inherent in life-course research. Routes of causation are entangled (Adler et al. 2012; Jones et al. 2011; Smith et al. 2010) by, for example, the correlations among childhood and adulthood poverty (Magnuson and Votruba-Drzal 2009), exposure to environmental toxins (Downey and Hawkins 2008), and chronic diseases (Profenno et al. 2010). One way to resolve these entanglements is to study the impact of exogenous shocks.

The Great Depression was a macroeconomic shock that exposed a large proportion of contemporary elders to stress and malnutrition when they were *in utero* through early childhood, a vulnerable period of neurocognitive development (cf. Benton 2010; Nyaradi et al. 2013). The shock of the Depression thereby functions as an exogenous shock to address two research questions: First, what is the impact on later-life cognitive function of early-life exposure to macroeconomic shock? Second, through what process does that exposure affect cognitive function? That is, does early-life exposure to macroeconomic shock affect later-life cognitive function because (1) hardship is

¹ Despite increasing awareness of false negative and false positive misdiagnoses of Alzheimer's disease, even with significant diagnostic error, Alzheimer's disease is the largest contributor to pathologic cognitive decline (Alzheimer's Association 2015; Qian et al. 2016; Skinner et al. 2016).

biologically embedded during the critical period of prenatal/early childhood (critical period model), and/or (2) sets an individual on a social trajectory for later-life triggers (social trajectory model), and/or (3) accumulates with other hardships over the life course (cumulative inequality model)?

The U.S. Health and Retirement Study (HRS) provides an opportunity to analyze cohort-level exposure to severe economic depression while considering individual-level characteristics. Based on the aforementioned theoretical models, I study how proposed risk factors have independent, cumulative, or multiplicative associations with later-life cognitive function. Policy implications of finding a long-term association between prenatal/early-life hardship and cognitive function include the need for social welfare programs that provide nutrition and support for pregnant people and young children. Furthermore, other interventions may be promising if intervening factors—such as education, exercise, and socialization—partially mitigate early-life harm.

Background and Hypotheses

Life-course theory provides a theoretical framework for understanding disparities in cognitive function. Drawing from sociology, demography, public health, and medical research, three theoretical models emerge to explain how life-course social disadvantage may affect later-life cognitive function. I briefly review the literature that motivates the theoretical models and describe how the use of natural experiments can provide analytical leverage.

Critical Period Models

Critical period models (CPM) (cf. Kuh et al. 2003) take as their object of study how hardship is written into one's physiology. There may be critical periods of development during which exposure to, for example, malnutrition or high stress causes irremediable damage to physiological systems (Hayward and Gorman 2004; Kajantie 2006; Lupien et al. 2009; McEwen and Gianaros 2011; Seeman et al. 2010). The effects of hardship are likely related to age, length, and extent of exposure (Manton et al. 1997).

As a type of CPM related to the prenatal period, Barker advanced through a series of studies (cf. 1990 and 1996) that fetal environment plays a role in the development of chronic diseases, independent of later-life risk factors—the fetal origins hypothesis. This prenatal period is understood as critical because it is a time of rapid growth and epigenetic plasticity (Barker 1990; Liu et al. 2010; Torche and Villarreal 2014). Prenatal exposure to a negative environment may actually cause the body to shift its *phenotype* (genetic expression) and/or cause functional or structural brain changes (McEwen 2009; Scholte et al. 2012).

Defining these critical periods is complex. Age of greatest neurological vulnerability varies by risk: for example, malnourishment, specific vitamin deficiencies, or stress (Benton 2010). More damage may result if adversity interrupts growth trajectories after they have begun than if there is hardship throughout the period. For example, evidence suggests if the first two trimesters *in utero* are characterized by higher nutritional inputs, which are then followed by deficiencies in the third trimester, the fetus may be at greater risk than if there were deficiencies through the entire fetal period (McEniry and

Palloni 2010). Indeed, the third trimester to age 2 may be the most sensitive period for neurological development because of the pace of brain development at these ages (Benton 2010; Casey et al. 2000; Mocerri et al. 2000). Between ages 3 to 6, cerebral volume reaches its maximum (Casey et al. 2000). This is a peak period of development in the frontal networks, critical to attention and planning (Thompson et al. 2000). Ages 7–10 appear to be critical for the development of the anterior corpus callosum, which is the region of the brain that declines the fastest in early stages of Alzheimer's (Thompson et al. 2000). Reduced brain volume and development are already detectable in adversity-exposed adolescents (Lupien et al. 2009).

However, confounding factors, such as prolonged poverty, have made CPMs challenging to evaluate (George 2005). Researchers have managed selectivity in the context of CPMs by using natural experiments. Part of the underlying logic of natural experiments is that the age at which a cohort experiences social changes is important to its level of sensitivity (Yang 2008). Researchers have established associations between morbidity and mortality and age during the Spanish flu epidemic, the Dutch famine, the German food crisis, the Chernobyl disaster, the Food Stamp Program rollout, and exposure to natural disasters (Almond 2006; Almond and Currie 2011; Almond et al. 2009, 2011; Conley and Bennett 2000; Jürges 2013; Lindeboom et al. 2010; McEniry and Palloni 2010; Torche 2011). Specific to cognition, macroeconomic conditions at birth appear to be associated with later-life cognitive function for European elders born in the first half of the twentieth century (Doblhammer et al. 2013; Fritze et al. 2014).

The Great Depression

The Great Depression is useful as an indicator of early-life conditions because of the breadth and the depth of the hardship experienced. Unemployment reached 27 % and even higher in some areas (Garcia 2014). With almost no federal assistance programs, approximately 60 % of the U.S. population met the federal guidelines for poverty, and one-fifth of all children in New York State and as high as 90 % of children in mining country were malnourished (Himmelgreen and Romero-Daza 2010). Because the Depression increased risk of malnutrition and allostatic load (physiological dysfunction related to stress) (cf. Geronimus et al. 2015; Juster et al. 2010; Seeman et al. 2010), it offers analytic leverage on whether early-life hardship is associated with later-life health. Although Cutler et al. (2007) found no significant long-term health effects of exposure to the Depression, they did not study cognitive function (further discussion in Online Resource 1).

Based on this body of research and using the Depression as an exogenous shock, I formulate a CPM hypothesis:

Hypothesis 1: Individuals who were at neurocognitively vulnerable ages (i.e., younger) during the Great Depression will have lower later-life cognitive function than those who had more years of neurocognitive development prior to the stock market crash.

For example, the cohort born during the Depression will have the lowest cognitive function scores compared with earlier cohorts, and the cohort aged 0–2 will have the next lowest scores (Benton 2010; Casey et al. 2000). The earliest cohort (born in 1909–

1918)—that is, the most neurocognitively developed at the onset of the Depression—will have the highest cognitive function scores. Evidence for the CPM hypothesis would be finding that intervening life-course factors do not significantly attenuate the association between early-life exposure to the Depression and cognitive function.

Social Trajectory Models

Nevertheless, deterministic arguments are seldom accurate in the social world. For instance, Elder (1998) found that by midlife, many Depression-era children were in good physical health and had overcome significant disadvantage. However, he also found that early adversity triggered a range of social transitions, including earlier marriages and antisocial behavior (Elder 1998). This is representative of a social trajectory model (STM) because he argued that the mechanism is sociobehavioral; Depression-era children possibly carry scars from that period that are not strictly physiological (Pol and Thomas 2002). In other words, it is not (necessarily²) that malnutrition or stress cause long-lasting physiological damage but rather that early hardship causes people to engage in riskier behaviors.

Do early-life experiences spur social trajectories that increase risk of cognitive dysfunction? The importance of distinguishing between physiological damage (CPM) and behavioral pathways (STM) relates to policy interventions. If cognitive function is associated with early-life hardship only through smoking behavior, for example, then investing in smoking-cessation programs may be the key intervention.

Hypothesis 2: Early-life exposure to the Great Depression increases the likelihood of engaging in risky behaviors over the life course (e.g., exercising less, smoking, drinking more alcohol).

Evidence for the STM hypothesis would be finding that prenatal or early-childhood exposure to the Depression is associated with riskier behaviors (identified as risky within the CPM).

Cumulative Inequality/Disadvantage Models

Although critical period models focus on measuring the independent effect of an exposure and the social trajectory model emphasizes behaviors, life-course theory requires us to consider “lives in motion,” (Elder 1998:7) examining entire life trajectories (Ben-Shlomo and Kuh 2002; Hayward and Gorman 2004). Most early-life conditions do not have direct, unalterable impacts on adulthood (Breteler 2001; Claussen et al. 2003; Mayer 2009).

Situated within life-course theory, cumulative inequality (Ferraro and Shippee 2009; Schafer et al. 2011, 2013) and cumulative disadvantage (DiPrete and Eirich 2006; Shuey and Willson 2008) theories posit that capital, including health, accumulates through the mechanisms of “structure, human agency, and chance” (Willson et al. 2007:1887). Regardless of the source of advantage, the advantaged have large initial

² Discerning whether they behave differently because of psychological or physiological changes is not possible here.

gains that accumulate over the lifetime, resulting in positive outcomes (Elder 1998; O’Rand and Hamil-Luker 2005; Pearlin et al. 2007; Shuey and Willson 2008). The converse is also true: those who are disadvantaged become increasingly disadvantaged.

However, social factors are not solely detrimental. Resilience is important to cognitive abilities, just as with general health (Schafer et al. 2013). Among autopsied patients, 10 % to 40 % exhibit Alzheimer’s-like brain pathology but appear asymptomatic (Meng and D’Arcy 2012). Derived from medical reserve and neuroplasticity theories, cognitive reserve (CR) is the latent construct that represents this gap between brain pathology and cognitive dysfunction (Meng and D’Arcy 2012; Stern 2012). Researchers have operationalized CR using several social factors but have relied heavily on formal education as a proxy. Education is considered not as it relates to SES or poverty’s toll on health but rather in its direct effect on the brain. A meta-analysis of CR research shows a strong association between education and Alzheimer’s diagnosis (OR = 2.62; $p < .001$) (Meng and D’Arcy 2012; Siegel 2012). Higher occupational attainment may also be associated with lower Alzheimer’s risk through CR (Stern 2012). Reed et al. (2011) found that later-life leisure cognitive activities were the most predictive of CR, net of education and occupation. Insights from this research, however, are limited because CR studies often do not control for confounding variables.

These factors that may build CR should be considered in cumulative inequality models (CIM) along with four disadvantage-related elements: onset, duration, magnitude of exposure, and interpretation or perception of risk (Ferraro and Shippee 2009; Schafer et al. 2011). People who experience repeated and/or severe hardship at vulnerable ages are at higher risk than those who experience a shorter period or lesser degree of harm, and their perception of that harm³ influences the extent of damage (Case and Paxson 2010; Torche 2011; Willson et al. 2007).

This research motivates the CIM hypothesis:

Hypothesis 3: Socioeconomic risk factors will accumulate over the life course, predicting multiplicatively worse later-life cognitive function and a faster rate of cognitive decline.

Whereas CPMs focus narrowly on irremediable harm caused during a critical moment—to the exclusion of other periods—CIMs do not preclude resilience and imply that the link between early life and health will be affected by intervening factors. Evidence for the CIM hypothesis would be if those who have low SES in early, mid-, and later life have worse cognitive function than those who have a shorter period of disadvantage.

Data and Methods

Data

Data are from 11 waves of the HRS, a longitudinal, population-based, nationally representative biennial survey ($n \approx 30,000$) covering a span of 20 years (1992–2012).

³ Unfortunately, there is no measure for perception of harm in the HRS, so this fourth factor cannot be tested.

The HRS is conducted by the University of Michigan (National Institute on Aging U01AG009740) (Health and Retirement Study 2014; RAND HRS Data 2014). In 1992, the HRS sampled two cohorts (born in 1890–1923 or 1931–1941) of noninstitutionalized individuals and their spouses (regardless of spouses' ages). The HRS continues to add cohorts to replenish the sample and to better represent the contemporary middle-aged and older population. The HRS uses both face-to-face and telephone interviews to survey early-life SES, occupation, later-life wealth, lifestyle, health, and cognitive function. I incorporate historical data from the U.S. Census Bureau, including regional birth rates, infant mortality, geographic mobility, and state-level unemployment rates. Restricting the data set to respondents born between 1909 and 1934 ($n = 14,481$), who participated in or after 1996 (when the cognition measures included in this study became consistent) ($n = 13,667$) and who had at least one cognitive function score, results in a final estimation sample of 12,278.

Dependent Variable: Fluid Cognition

The HRS adapted the Telephone Interview for Cognitive Status (TICS) to evaluate cognitive deficits and dementia-related declines (Ofstedal et al. 2002). The TICS has near-normal distribution and high test-retest reliability, is relatively insulated against ceiling effects, and effectively predicts cognitive impairment (Crimmins et al. 2011; Fong et al. 2009; Karlamangla et al. 2009). The University of Michigan Survey Research Center used a multivariate, regression-based procedure to impute missing, refusal, or not-applicable responses to cognition questions for nonproxied respondents who completed an interview that wave (for details, see Fisher et al. 2017).

From TICS, I distinguish fluid from crystallized intelligence because they are distinct aspects of cognition and show different life-course trajectories (Harvey et al. 2003; Horn 1982; van den Berg et al. 2010). *Fluid intelligence* is based in problem-solving ability, develops early, follows a normative decline, and is less correlated with education (Ghisletta et al. 2012). *Crystallized intelligence* is related to educational and cultural factors (e.g., vocabulary), develops later in life, and is more stable (Ghisletta and Lindenberger 2004; Ghisletta et al. 2012). This analysis focuses on fluid cognition because it is more reflective of neurophysiological health (Buckner 2004; Ghisletta et al. 2012).

Using TICS in Waves 3–11⁴ (1996–2012), I generate separate standardized scores for three items: (1) total word recall, (2) counting backward from 20, and (3) serial sevens (Cook et al. 2009). These measures are administered upon survey entry and then at every wave thereafter (biennially) unless the respondent requires a proxy. *Total word recall* is the sum of immediate and delayed word recall, wherein respondents recall immediately and after five minutes 10 words from one of four lists (RAND HRS Data 2014). *Counting backward* is denoted as 2 = correctly counts backward for 10 continuous numbers on the first try, 1 = successful on the second try, 0 = unsuccessful. *Serial sevens* is scored as 0–5, equaling the number of times the respondent correctly subtracts seven starting from 100 (e.g., 5 = five correct subtractions). Principal components analysis validates combining the three standardized scores into a single variable. I average the three standardized scores to generate fluid cognition.

⁴ I exclude the first two waves because some TICS measures were administered inconsistently.

Independent Variables

Cohort I construct *Cohort* based on when the respondent was born relative to the Depression, corresponding to the periods of neurological vulnerability outlined in the Background and Hypotheses section (Lupien et al. 2009; Thompson et al. 2000). The Great Depression cohort⁵ was born January 29, 1930–1934;⁶ thus, their last trimester *in utero* (approximately) and birth was during the Depression. I hypothesize they are the most vulnerable cohort (Doblhammer et al. 2013; Lupien et al. 2009). The next cohort was age 2 and under when the stock market crashed (born in 1927 to January 28, 1930). The other cohort divisions are constructed to demarcate successively more insulative years of neurocognitive development prior to the stock market crash (aged 3–6, born in 1923–1926; aged 7–10, born in 1919–1922; and over age 10, born in 1909–1918).

Demographic Factors Cognitive function is nonlinear over the life course, so I center age at 60 and include a quadratic age term. HRS records binary *Sex* (1 = female). I generate a *Race/Ethnicity* variable: non-Hispanic white, black/African American and Hispanic black, nonblack Latinx, and other.⁷

Early SES I control for *Childhood Region*⁸ because social factors, such as schooling quality, vary regionally (Glymour et al. 2008). To paint a more comprehensive picture of childhood circumstances and mitigate concerns about recall bias,⁹ I include multiple early-life SES measures: binary indicators for *Rural* childhood; *Father's Economic Contribution* (1 = unemployed/absent/deceased); self-reported *Family SES* (poor vs. average/wealthy); *Childhood Health* (poor/fair or good/very good/excellent); and *Parents' Education*, which is average of mother's and father's education¹⁰ (<1, 1–6, 6.25–8.75, 9–11.5, 12+).

Educational Attainment is a categorical variable (less than high school, high school/general educational development (GED), some college, and college or more) to account for the nonlinear association between education and mortality (Montez et al. 2012).

Mid- to Later-Life SES¹¹ I collapse RAND's *Longest Occupation* categories (as of last wave of participation) into the following: office (office, professional, and government work); armed forces; service; manual (construction, extraction, and manufacturing); and

⁵ The Great Depression cohort is not synonymous with the HRS category *Children of the Depression Age*.

⁶ I set the upper range of 1934 because those born toward the end of and after the Depression, which officially ended in 1941, are too young for their data to be used to accurately estimate age-related cognitive decline.

⁷ "Black-Hispanics" may have more similar health outcomes to blacks than to nonblack Hispanics (Elo et al. 2008).

⁸ I substitute birthplace for childhood region when region is missing. In 1890, 95.5 % of people lived in the region in which they were born; that remained above 90 % until 1930 (still 87 %) (U.S. Bureau of the Census 1975). More than 90 % of HRS respondents with nonmissing data have the same childhood region as their birthplace.

⁹ Although retrospective reports of early-life SES and health could be affected by current health status or other recall biases, an evaluation of a subset of HRS respondents showed high internal consistency between self-reported early-life health and long-term childhood health limitations (Elo 1998). I also conduct robustness checks, excluding respondents who are cognitively impaired when they first take the TICS (see Online Resource 1 for details on the sensitivity analyses).

¹⁰ Where father's education is missing, mother's stands alone ($n = 554$).

¹¹ In these HRS cohorts, education, wealth, and occupation are not too strongly correlated to be studied independently (Glymour 2007).

farming, forestry, and fishing. To capture later-life wealth versus fluctuations related to retirement, market volatility, and/or gifting, I average the RAND *Wealth* measure (income plus assets minus debt) over the survey period (in debt; \$0–49,999; \$50,000–199,999; \$200,000–499,999; \$500,000–999,999; \$1 million or more).

Life-Course Measures *Early SES* and quartiles thereof are generated using factor analysis combining father's economic contribution, parents' education, self-reported childhood SES and health, and education (no degree, GED, high school diploma, associate degree/some college, bachelor's, master's, law/MD/PhD) (eigenvalue = 1.87). *Life-course SES* and quartiles thereof include the same measures of early-life SES, plus *Occupation* and *Wealth* (eigenvalue = 2.36).

Behavioral Factors Covariates include *Partnership* (never married, widow(er)ed, separated/divorced/spouse absent, or partnered/married) and binary indicators for *Socializing With Neighbors* (daily–monthly, yearly–never), *Exercising* once per week or less, and *Currently Smokes*. *Alcohol Use* is typical consumption over the last three months—abstinent or rare, 1–2 drinks/day, or 3+ drinks/day. All behavioral factors are time-variant, and *Socializing* and *Exercising* are lagged by one wave.

Health Factors *BMI* is a time-invariant categorical variable of average BMI over the study period¹² (Centers for Disease Control and Prevention thresholds). *CESD* is respondent's score on the Center for Epidemiologic Studies Depression Scale (CESD) (0, 1, 2–4, 5–8; higher scores = reporting more depressive feelings). The *Comorbidity Index* (0–4) is self-reported doctor diagnosis for stroke, diabetes/high blood sugar, a heart condition, and/or high blood pressure/hypertension. *CESD* and the *Comorbidity Index* are time-variant and lagged by one wave.

Controls *Proxy Attrition* and *Death Attrition* indicate whether a respondent needed a proxy or died between 1992 and 2012.

Missingness from rolling enrollment is plausibly missing at random (MAR), but withdrawal due to morbidity or mortality violates the MAR assumption; thus, multiple imputation is not appropriate. Childhood rurality, parents' education, and occupation¹³ account for most time-invariant missingness. Fluid cognition is statistically significantly higher for those who have complete data ($b = 0.45, p < .001$). For these reasons, I retain a missingness category for each categorical variable.

Analytic Strategy

To estimate *Fluid Cognition* testing the CPM and CIM hypotheses, I use growth curve models, which produce trajectories of cognitive change as a function of the predictors (Leahey and Guo 2001; Raudenbush and Bryk 2002). Individuals' scores contribute to

¹² *Average BMI* captures a pattern of weight and is thus more indicative of general health and less susceptible to reverse causation.

¹³ Women account for 78 % of those missing the occupation variable. There is no category for “homemaker/working in the home.”

estimates even if there are few observations; thus, these models are more robust to rolling enrollment and attrition (Curran et al. 2010). Approximately 75 % of the sample has three or more observations of fluid cognition. These growth curve models are two-level hierarchical models with individuals at Level 2, individuals' multiple fluid cognition scores at Level 1, and a random coefficient for *Cohort*:

$$Y_{it} = \alpha + \beta_{1i}Age_{it} + \beta_{2i}Cohort_i + \beta_{3i}(Age_{it} \times Cohort_i) + \sum_{j=1}^m \beta_{wj}w_{ij} + \sum_{k=1}^p \beta_{xk}x_{itk} + u_i + \varepsilon_{it} . \quad (1)$$

Fluid cognition (Y_{it}) is a function of the intercept α (the grand mean of all respondents' cognitive tests), *Age* and *Quadratic Age* slopes (β_1 and β_2), *Cohort* (β_3), sets of time-invariant (w) predictors for person i and time-variant (x) predictors for person i at age t , the person-specific error term (μ), and the overall model residual (ε). The intercept parameter α can be interpreted as the mean standardized fluid cognition score at age 60.

To test the CPM hypothesis, I use a staged life-course approach to estimate nested models. For the STM hypothesis, I estimate multilevel binary and multinomial logistic and linear regression models to examine whether exposure to the Depression is associated with behavioral risk factors. To test the CIM hypothesis, I include interactions of life-course factors. I also predict marginal values of fluid cognition, displaying results graphically to aid in interpretation. I use clustered standard errors within persons in Stata 14. All results from analyses not displayed are available upon request.

Results

I present my results in several steps, starting with descriptive statistics by cohort. Subsequent tables present results of the regression models for the CPM, STM, and CIM hypotheses. For time-variant statistics, I display the mean of the worst value recorded over the period—for example, the highest number of comorbidities for person i at any age t (Table 1).

The mean fluid cognition scores are statistically significantly lower for the earliest cohort. Parents' likelihood of attending high school or more increases dramatically across the cohorts. The most recent cohort is significantly more likely to graduate college and work in office/professional or service occupations. Compared with the two more recent cohorts, the earliest cohorts have significantly lower life-course SES but higher rates of emotional depression and widow(er)hood. The more recent cohorts are more likely to be separated/divorced or have an absent spouse, to socialize with neighbors yearly or hardly ever, to currently smoke, and to have higher BMIs.

Hypothesis 1: Critical Period Model

I consider the CPM to investigate whether prenatal and/or early-childhood exposure to macroeconomic shock has an independent association with fluid cognition, regardless of other life-course factors. Across all four nested models, compared with the reference cohort (over age 10 at the onset of the Depression), I find that cohorts born during or closer to the Depression have statistically significantly lower fluid cognition (Table 2).

Table 1 Descriptive statistics by cohort

	1909–1918		1919–1922		1923–1925		1926–1/28/1930		1/29/1930–1934	
	Mean %	SD	Mean %	SD	Mean %	SD	Mean %	SD	Mean %	SD
Persons	2,815		2,035		2,045		1,768		3,615	
Fluid Cognition ^a	-0.43	0.79	-0.24	0.75	-0.17	0.72	-0.07	0.69	-0.00	0.70
Demographic Characteristics ^a										
Age	85.3	4.8	80.7	5.0	78.0	4.9	75.1	4.9	70.6	5.3
Sex (1 = female)	62.5	0.5	58.6	0.5	54.2	0.5	51.8	0.5	54.7	0.5
Race/ethnicity										
White	81.1		80.0		82.1		81.4		74.3	
Black	12.7		13.2		9.9		10.5		15.9	
Latinx, nonblack	4.9		5.7		6.2		6.0		7.9	
Other	1.3		1.1		1.8		2.2		1.9	
Childhood Region ^a										
New England	5.9		5.5		6.2		5.7		5.0	
Mid-Atlantic	18.2		18.2		15.2		15.6		14.5	
Midwest-East North Central	20.3		19.1		19.9		19.1		16.1	
Midwest-West North Central	9.9		10.1		10.9		11.6		9.6	
South Atlantic	14.5		14.9		15.0		14.0		17.6	
East South Central	5.9		7.2		7.4		6.7		9.1	
West South Central	12.1		10.0		8.7		9.5		9.6	
West Mountain	2.1		2.5		2.8		3.3		3.6	
West Pacific	5.0		6.3		7.1		7.1		6.1	
State Missing	0.0		0.2		0.1		0.0		0.0	

Table 1 (continued)

	1909–1918		1919–1922		1923–1925		1926–1/28/1930		1/29/1930–1934	
	Mean %	SD	Mean %	SD	Mean %	SD	Mean %	SD	Mean %	SD
Non-U.S. state	6.1		6.1		6.7		7.5		8.7	
Childhood Rural ^a	40.0	0.5	42.0	0.5	46.0	0.5	46.0	0.5	47.0	0.5
Early-Life SES ^b										
Father no job	29.3	0.5	35.2	0.5	34.2	0.5	33.5	0.5	29.9	0.5
Family poor	33	0.5	36.9	0.5	36.2	0.5	35.4	0.5	33.8	0.5
Health good+	92.5	0.3	94.1	0.2	93.9	0.2	94.3	0.2	93.2	0.3
Parents' education										
<1	0.4		0.6		2.4		3.1		2.7	
1–6	1.1		1.9		11.0		14.5		18.2	
6.25–8.75	90.5		89.0		52.0		31.5		25.7	
9–11.5	0.4		1.0		9.0		17.6		20.6	
High school+	0.8		1.4		14.1		20.5		23.7	
Educational Attainment ^a										
Less than high school	43.0		33.4		32.6		28.1		28.1	
High school	32.0		35.7		34.3		37.3		36.8	
Some college	15.2		17.3		18.1		17.0		18.0	
College+	9.9		13.6		15.0		17.6		17.2	
Occupation ^a										
Office/professional	34.7		39.9		43.6		46.4		47.8	
Armed forces	0.0		0.0		0.2		0.6		1.0	
Manual	21.8		21.1		22.0		23.0		23.4	

Table 1 (continued)

	1909–1918		1919–1922		1923–1925		1926–1/28/1930		1/29/1930–1934	
	Mean %	SD	Mean %	SD	Mean %	SD	Mean %	SD	Mean %	SD
Service	8.8		8.3		9.6		10.0		13.3	
Farm/forest/fish	3.8		3.5		3.5		2.5		3.1	
Wealth ^a										
In debt	1.6		2.4		1.4		2.0		2.6	
\$0–\$49,999	29.1		24.6		19.7		19.0		21.3	
\$50K–\$199,999	28.6		27.7		30.4		30.2		31.3	
\$200K–\$499,999	17.1		20.7		24.9		25.9		24.7	
\$500K–\$999,999	6.5		9.9		12.7		13.4		12.2	
\$1 million+	4.6		6.0		7.7		9.1		7.7	
Life-Course SES ^a	-0.36	0.8	-0.24	0.9	-0.1	0.9	-0.05	1.0	-0.1	1.0
Behavioral Factors										
Partnered/married ^a	0.8		3.8		13.3		21.4		27.6	
Never married	0.2		0.4		0.8		0.9		1.8	
Widow(er)ed	7.3		16.4		20.6		20.2		19.2	
Absent	0.6		1.6		2.6		4.1		5.6	
Social ≥ monthly ^a	39.6	0.5	38.8	0.5	37.6	0.5	37.2	0.5	33.0	0.5
Exercise ≤ 1/week ^a	94.0	0.2	93.0	0.3	92.0	0.3	89.0	0.3	93.0	0.3
Alcohol Use ^a										
Abstinent/rare	69.6		60.1		57.7		55.2		53.2	
1–2 drinks/day	25.9		33.2		33.8		33.5		32.1	
3+ drinks/day	4.4		6.7		8.5		11.4		14.6	

Table 1 (continued)

	1909–1918		1919–1922		1923–1925		1926–1/28/1930		1/29/1930–1934	
	Mean %	SD	Mean %	SD	Mean %	SD	Mean %	SD	Mean %	SD
Smokes ^a	8.0	0.3	11.0	0.3	14.0	0.3	17.0	0.4	20.0	0.4
Physical Health										
Average BMI ^a										
Underweight	4.2		2.4		2.0		1.5		1.2	
Normal	53.5		45.0		41.4		34.7		32.1	
Overweight	32.3		39.4		40.1		42.5		42.1	
Obese	9.5		12.6		15.5		19.8		22.1	
≥40	0.3		0.5		1.0		1.4		2.2	
Emotional depression ^a										
0	9.0		9.0		11.3		12.8		13.1	
1	14.5		18.3		20.5		21.3		20.0	
2–4	45.7		44.2		41.8		39.5		39.3	
5–8	30.8		28.5		26.1		26.1		27.5	
Comorbidities ^a										
Zero	12.0		12.2		13.7		12.4		14.3	
One	33.0		29.5		30.6		31.0		32.5	
Two	34.2		35.5		33.3		34.6		31.7	
Three	17.8		18.6		18.0		16.8		17.5	
Four	3.0		4.2		4.4		5.2		4.1	

Note: Categories for missingness are excluded for presentation purposes but are included in all analyses.

^a Differences among cohorts statistically significant at $p < .001$.

Table 2 Critical period model: Selected coefficients from nested growth curve models

	(1) Demographic		(2) Early-Life SES		(3) Education		(4) Full Model	
	<i>b</i>	SE	<i>b</i>	SE	<i>b</i>	SE	<i>b</i>	SE
Cohort (ref. = 1909–1918)								
1919–1922	-0.073***	0.017	-0.102***	0.016	-0.117***	0.019	-0.119***	0.016
1923–1925	-0.161***	0.018	-0.213***	0.017	-0.186***	0.021	-0.202***	0.017
1926–1/28/1930	-0.177***	0.018	-0.252***	0.019	-0.225***	0.024	-0.231***	0.019
1/29/1930–1934	-0.231***	0.017	-0.308***	0.019	-0.280***	0.033	-0.292***	0.019
Sex (female = 1)	-0.023*	0.010	-0.016†	0.009	-0.001	0.009	0.016	0.010
Race/Ethnicity (ref. = white)								
Black	-0.641***	0.015	-0.491***	0.015	-0.438***	0.015	-0.379***	0.015
Latinx, nonblack	-0.496***	0.020	-0.344***	0.023	-0.250***	0.022	-0.194***	0.022
Other	-0.334***	0.038	-0.265***	0.037	-0.255***	0.035	-0.209***	0.035
Childhood Rural (ref. = city/town)								
Father Unemployed/Absent/Dead			-0.127***	0.010	-0.073***	0.010	-0.068***	0.010
Family Poor			-0.033**	0.011	-0.017†	0.010	-0.013	0.010
Childhood Health Good+			-0.015	0.011	0.012	0.010	0.017†	0.010
Parents' Education (ref. = <1)			0.087***	0.019	0.067***	0.018	0.050**	0.018
1–6			0.299***	0.037	0.258***	0.035	0.236***	0.034
6.25–8.75			0.403***	0.036	0.302***	0.034	0.270***	0.033
9–11.5			0.446***	0.037	0.305***	0.035	0.268***	0.034
High school+			0.470***	0.037	0.287***	0.035	0.254***	0.034
Education (ref. = high school/GED)								
Less than high school					-0.308***	0.012	-0.255***	0.012
Some college					0.098***	0.013	0.060***	0.013
College+					0.193***	0.014	0.111***	0.015

Table 2 (continued)

	(1) Demographic		(2) Early-Life SES		(3) Education		(4) Full Model	
	<i>b</i>	SE	<i>b</i>	SE	<i>b</i>	SE	<i>b</i>	SE
Occupation (ref. = office/professional)								
Armed forces			0.031				0.031	0.060
Manufacturing/construction/extraction			-0.094***				-0.094***	0.013
Service			-0.128***				-0.128***	0.016
Farm/forest/fish			-0.141***				-0.141***	0.026
Wealth (ref. = \$0-\$49,999)								
In debt							-0.048	0.032
\$50K-\$199,999							0.127***	0.013
\$200K-\$499,999							0.162***	0.014
\$500K-\$999,999							0.172***	0.018
\$1 million+							0.215***	0.021
Partnership Status (ref. = partnered/married)								
Never married							-0.026	0.024
Widow(er)ed							0.031***	0.007
Separated/divorced/absent							0.021 [†]	0.012
Socializing Daily/Monthly (ref. = yearly/never)							0.019***	0.006
Exercise 1/Week or Less							-0.018***	0.005
Alcohol Use (ref. = 1-2 drinks/day)								
Abstinent/rare							-0.034***	0.007
3+ drinks/day							-0.032*	0.013
Currently Smokes							-0.001	0.011

Table 2 (continued)

	(1) Demographic		(2) Early-Life SES		(3) Education		(4) Full Model	
	<i>b</i>	SE	<i>b</i>	SE	<i>b</i>	SE	<i>b</i>	SE
BMI (ref. = normal)								
Underweight (≤18.5)							-0.003	0.032
Overweight, (25–29)							0.014	0.010
Obese (30–39)							0.038**	0.013
≥40							0.065†	0.039
Emotional Depression (CESD) (ref. = 0)								
1							0.002	0.006
2–4							-0.021***	0.006
5–8							-0.041***	0.009
Comorbidity Index (ref. = two)								
None							0.029***	0.008
One							0.022**	0.007
Three							-0.048***	0.010
Four							-0.095***	0.026
Intercept	0.686***	0.022	0.358***	0.049	0.405***	0.047	0.377***	0.050
Persons	12,278		12,278		12,278		12,278	
Person-Years	62,756		62,756		62,756		62,756	
Chi-squared	12,848		14,708		16,476		17,531	

Note: Values are adjusted for item missingness and all variables. Complete coefficients and the random effects parameters are presented in Online Resource 1.

† $p < .10$; * $p < .05$; ** $p < .01$; *** $p < .001$

Later life-course factors do not significantly attenuate the exposure coefficient—evidence for the CPM hypothesis.

In the full model, those hypothesized to be most vulnerable—that is, the Great Depression cohort—have the worst fluid cognition compared with the 1909–1918 cohort ($b = -0.292$, $p < .001$), net of all other factors. The fewer years of cognitive development a cohort had prior to the stock market crash, the worse their predicted fluid cognition compared with the reference cohort (aged 0–2, 3–6, and 7–10, respectively; $b = -0.231$, $b = -0.202$, $b = -0.119$, $p < .001$). The cohort coefficient magnitudes are comparable to or greater than those for education, which has been the most significant social predictor found to date (cf. Meng and D’Arcy 2012).

To further contextualize the cohorts’ cognitive disparities, at age 80, the Great Depression cohort’s fluid cognition is almost one-half standard deviation below the mean of 0, whereas the earliest cohort’s predicted score remains slightly below 0, net of other factors (see Fig. 1). By comparison, elders who have already been diagnosed with a memory disease have a mean fluid cognition of -0.9 , only slightly lower than the Great Depression cohort’s fluid cognition at age 80.

Interactions between cohort and the age terms are nonsignificant. However, because they started with lower fluid cognition, the Great Depression cohort is predicted to reach any given cognitive impairment threshold 5 to 10 years prior to the earliest cohort (Fig. 1). The Great Depression cohort’s fluid cognition at age 75 is worse than the 1909–1918 cohort’s fluid cognition at age 82.

After education is controlled, sex is not significant. Although the associations between race/ethnicity and fluid cognition in the full model are attenuated by approximately one-half, the fluid cognition for people of color remains significantly lower than that of whites’ (black $b = -0.379$, Latinx $b = -0.194$, other $b = -0.209$, $p < .001$). The Latinx-white cognitive disparity is the most attenuated by other life-course factors (60 %). Childhood rurality is negatively associated with fluid cognition. Better early-life health, higher parents’ and respondents’ education, higher occupational attainment, greater later-life wealth, and socializing more frequently with neighbors are associated with higher fluid cognition. Widow(er)hood and separated/divorced/absent spouse are associated with higher cognitive function than partnered/married.¹⁴ Exercising more frequently, light alcohol use (reference = abstinent/rare), higher average BMI (reference = normal), lower emotional depression, and fewer comorbidities are associated with higher fluid cognition.

Hypothesis 2: Social Trajectory Model

As noted, several behavioral factors are predictive of later-life cognitive function (Table 2). Is it possible that early-life harm increases the probability of engaging in “risky behaviors” over the life course, which subsequently increases the risk of later-life cognitive dysfunction? Here I examine whether behavioral factors¹⁵ are associated with

¹⁴ Because this is inconsistent with the literature, I explore this association. Interacting partnership/marital status with sex shows that only women in these categories have higher cognitive function. In the full data set ($n = 33,393$) using the CPM, those in the never-married category have statistically significantly lower fluid cognition, but the differences among the other categories are nonsignificant. However, men’s fluid cognition is more negatively affected by widowhood and being separated/divorced/absent than women’s.

¹⁵ Table S3 (Online Resource 1) depicts sensitivity analyses testing cohort’s association with nonbehavioral risk factors (e.g., stroke).

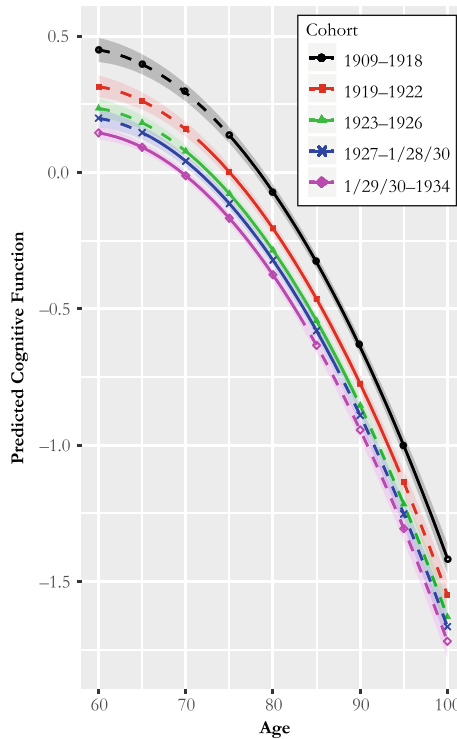


Fig. 1 Predicted fluid cognition by cohort over age. Solid lines indicate ages represented in the data (95 % confidence intervals)

exposure to macroeconomic shock, early-life poverty, and their interaction, controlling for age, sex, and race/ethnicity.

Contrary to expectation, the interactions between cohort and early-life SES are primarily nonsignificant (results available upon request). For example, respondents from the Great Depression cohort who had lower SES in childhood are no more likely to have lower occupational attainment than those who had lower SES in the 1909–1918 cohort.

I highlight several of the STM outcomes to demonstrate cohort differences in risky behaviors (see Table 3 and Fig. 2). Net of controls, those in the Great Depression cohort are significantly more likely than the three earliest cohorts (born before 1927) to have a high school diploma versus less than high school. Compared with the earliest cohort (born in 1909–1918), the Great Depression cohort was significantly more likely to attend college (vs. high school), to be wealthier, to work in professional/office occupations (vs. service/manual), to be light drinkers (vs. abstinent/rare), and to exercise more than once per week (vs. once/week or less). The CPM suggests that these factors are all associated with higher fluid cognition. However, the Great Depression cohort is significantly more likely than the earliest and neighboring cohort to consume three or more alcoholic drinks/day, which is associated with lower fluid cognition. There is not a clear, significant pattern by partnership status or socializing that would indicate the Great Depression cohort is at either higher or lower risk. Differences between the Great Depression cohort and the neighboring cohorts are primarily nonsignificant.

Table 3 Social trajectory model: Predicting life-course factors by cohort and early-life SES

		Predictors:																	
		1909–1918			1919–1922			1923–1926			1927–1/28/1930			Early-Life SES			Constant		
Outcomes	<i>b</i>	SE	<i>b</i>	SE	<i>b</i>	SE	<i>b</i>	SE	<i>b</i>	SE	<i>b</i>	SE	Coef.	SE	Coef.	SE	Coef.	SE	
Education (ref. = high school/GED)																			
Less than high school	0.86***	0.07	0.33***	0.08	0.31***	0.08	-0.02	0.09	-0.41***	0.03	-0.81***	0.06							
Some college	0.06	0.08	0.04	0.09	0.13	0.09	-0.03	0.09	0.32***	0.03	-0.56***	0.06							
College+	-0.26**	0.09	-0.01	0.09	-0.04	0.09	0.02	0.09	0.73***	0.04	-0.35***	0.06							
Occupation (ref. = office/professional)																			
Service/manual	0.31***	0.07	-0.00	0.07	0.02	0.07	-0.07	0.07	-0.39***	0.03	-0.28***	0.05							
Wealth (ref. = \$50K–\$199,999)																			
In debt	-0.17	0.22	0.28	0.21	-0.43	0.26	0.04	0.24	-0.26**	0.08	-3.73***	0.20							
\$0–\$49,999	0.64***	0.08	0.38***	0.09	0.09	0.09	0.06	0.10	-0.11***	0.03	-1.15***	0.07							
\$200K–\$499,999	-0.37***	0.08	-0.06	0.08	0.00	0.08	0.06	0.09	0.25***	0.03	0.11 [†]	0.06							
\$500K–\$999,999	-0.68***	0.11	-0.16	0.11	0.03	0.10	0.07	0.10	0.39***	0.04	-0.42***	0.07							
\$1 million+	-0.55***	0.12	-0.09	0.13	-0.03	0.12	0.12	0.12	0.72***	0.05	-0.86***	0.08							
Partnership Status (ref. = partnered/married)																			
Never married	0.02	0.20	-0.23	0.20	-0.53*	0.21	-0.53*	0.22	-0.09	0.08	-3.96***	0.18							
Widow(er)ed	0.34***	0.07	0.23***	0.07	-0.17*	0.07	-0.25**	0.08	-0.05 [†]	0.03	-3.38***	0.07							
Separated/divorced/absent	-0.30*	0.12	-0.39***	0.12	-0.73***	0.12	-0.43***	0.12	-0.05	0.04	-2.92***	0.09							
Socializing Less	0.00	0.06	-0.07	0.05	-0.08	0.05	-0.02	0.05	0.10***	0.02	-1.23***	0.04							
Exercise >1/week	-0.74***	0.05	-0.38***	0.05	-0.21***	0.04	-0.08 [†]	0.04	0.10***	0.02	0.38***	0.04							

Table 3 (continued)

Outcomes		Predictors: Cohort (ref. = Great Depression cohort born 1/29/1930–1934)												
		1909–1918		1919–1922		1923–1926		1927–1/28/1930		Early-Life SES		Constant		
	<i>b</i>	SE	<i>b</i>	SE	<i>b</i>	SE	<i>b</i>	SE	<i>b</i>	SE	Coef.	SE	Coef.	SE
Alcohol Use (ref. = abstinent/rare)														
1–2 drinks/day	–0.34***	0.07	–0.04	0.07	–0.01	0.07	0.03	0.07	0.29***	0.02	–0.40***	0.05		
3+ drinks/day	–0.50**	0.17	–0.19	0.15	–0.19	0.15	–0.30*	0.14	0.10 [†]	0.05	–1.08***	0.08		
Currently Smokes	0.19	0.13	0.22 [†]	0.12	0.12	0.11	0.13	0.10	–0.03	0.04	–1.25***	0.07		

Note: All models are adjusted for sex, race/ethnicity, and item missingness; time-variant outcomes are also adjusted for age.

[†] $p < .10$; * $p < .05$; ** $p < .01$; *** $p < .001$

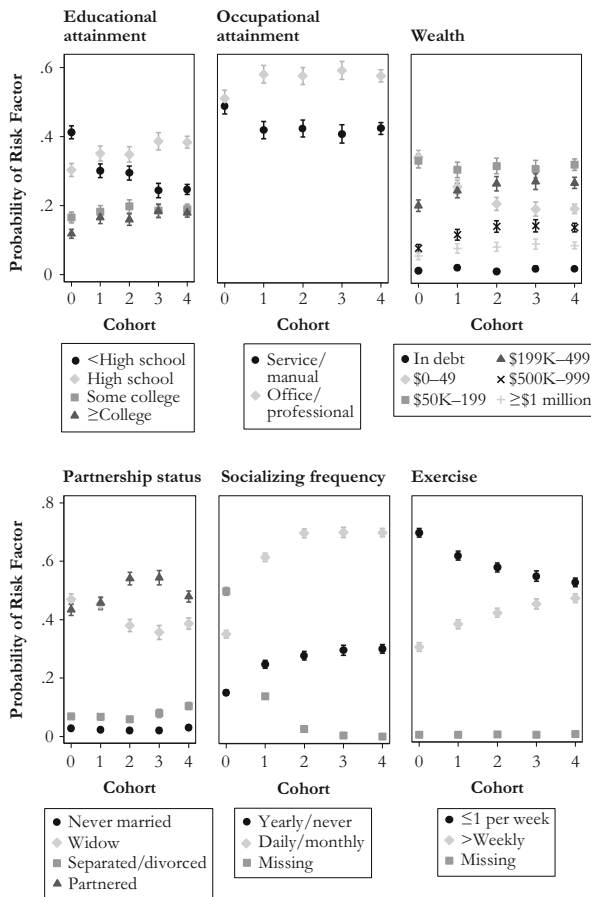


Fig. 2 Social trajectory model: Predicted probabilities of risk factors by cohort, net of sex, race/ethnicity, and early-life SES. Time-variant outcomes are also adjusted for age

In sum, there is little evidence that the cohorts exposed to the Depression at earlier ages engage in riskier behaviors, net of controls. The STM hypothesis is not supported. The mechanism connecting early-life exposure to the Great Depression with fluid cognition does not appear to be behavioral.

Hypothesis 3: Cumulative Inequality Model

To test whether life-course factors interact to affect fluid cognition and rate of cognitive decline, I estimate models interacting age, cohort, and early-life SES with other factors. In regard to the age interactions, few factors significantly predict rate of decline, so I do not display these results. That said, it is notable that blacks have a faster linear rate of fluid cognition decline than whites (race/ethnicity main effect: $b = -0.27$, interaction with age: $b = -0.01, p < .001$). Aligned with prior research (Early et al. 2013; Wilson et al. 2005), education and rate of decline are only weakly associated.

Similarly, little insight is gained from the interaction of cohort and life-course factors, although lower education does predict significantly worse fluid cognition for

the two most recent cohorts compared with the 1909–1918 cohort (results available upon request). Contrary to my hypothesis, the interaction between cohort and early-life SES is nonsignificant. For instance, respondents in the Great Depression cohort who were from lower-SES families do not have multiplicatively worse fluid cognition, net of later-life factors (Table 4).

In contrast, later-life factors do affect the association between early-life SES and fluid cognition (Table 4). Fluid cognition is multiplicatively lower if an individual has lower early-life SES and lower occupational attainment or lower later-life wealth (Fig. 3). If an individual was in the lowest quartile of early-life SES, being in debt in later life predicts fluid cognition approximately three-quarters of a standard deviation lower than had s/he started in the highest quartile of early-life SES (fluid cognition = -0.74 vs. -0.14). The cognitive disparity by occupational attainment and wealth is narrower for those with higher early-life SES, net of other factors (Fig. 3). These results suggest that (1) higher early-life SES is protective against later-life risk factors for cognitive decline, and/or (2) midlife SES mitigates the negative effects of lower early-life SES.

I next predict fluid cognition for those who had a lifetime of low, mixed, or high SES, using estimates from the full CPM. Based on Table 2, the low-SES factors are Great Depression cohort, low parental education, poor childhood health, less than high school, in debt, and service occupations. The high-SES or mitigating factors are the most beneficial alternative category. I divide life into “early” and “late” after respondent’s education; control for age terms, proxy, death, partnership (married), and childhood region (Mid-Atlantic); and hold all other variables except race/ethnicity and sex at their means. Figure 4 displays predicted fluid cognition for blacks, Latinx, and white men and women at age 75.

Low life-course SES results in the lowest fluid cognition—more than 1 standard deviation below the higher-SES predictions (Fig. 4). We can interpret Fig. 4 to indicate that low early-life SES is worse for fluid cognition than low later-life SES. Notably, blacks and Latinx have significantly worse fluid cognition than whites across all groups. At age 75, blacks who have low SES throughout their lives have a predicted fluid cognition over 1 standard deviation below the mean, comparable with those who already have a memory disease diagnosis (-0.9). Although racial/ethnic differences are attenuated in the high life-course SES scenario, disparities remain.

Sensitivity Analyses

I conduct multiple sensitivity analyses.¹⁶ The selection and attrition that are challenges for most longitudinal surveys are here exacerbated by the HRS’s rolling enrollment and aged sample. To account for selection into the sample, I restrict analyses to ages 76–83 or 72–83. Results are consistent (Online Resource 1, Fig. S1). In all models, I address the issue of selective mortality biasing cohort estimates by including indicators for whether respondents ever needed a proxy or died in 1992–2012.

As another robustness check, I vary cohort construction. I define the “most-exposed” cohort as under age 2 at the stock market crash or born during the Depression (born in 1927–1934; $b = -0.26$, $p < .001$), or conceived after the stock market crash (born in

¹⁶ Results are available upon request.

Table 4 Cumulative inequality models: Regressing fluid cognition on interactions of early-life SES with cohort, occupation, and wealth

	Model 1. Early-Life SES × Cohort		Model 2. Early-Life SES × Occupation		Model 3. Early-Life SES × Wealth	
	<i>b</i>	SE	<i>b</i>	SE	<i>b</i>	SE
Early-Life SES	0.10***	0.016	0.04***	0.008	0.17***	0.012
Cohort (ref. = 1909–1918)						
1919–1922	-0.10***	0.019	0.04	0.066	-0.02	0.051
1923–1925	-0.20***	0.019	-0.18***	0.015	0.12***	0.016
1926–1/28/1930	-0.22***	0.021	-0.18***	0.020	0.18***	0.017
1/29/1930–1934	-0.30***	0.020	-0.22***	0.032	0.20***	0.019
Occupation (ref. = office)						
Armed forces						
Manual						
Service						
Farm/forest/fish						
Early-Life SES × Occupation						
Armed forces	0.02	0.022	-0.12	0.082	0.05	0.045
Manual	-0.01	0.020	0.08***	0.015	-0.08***	0.016
Service	-0.01	0.020	0.11***	0.020	-0.10***	0.016
Farm/forest/fish	0.00	0.018	0.15***	0.031	-0.12***	0.020
Early-Life SES × Cohort						
1919–1922						
1923–1925						
1926–1/28/1930						
1/29/1930–1934						
Early-Life SES × Adult Wealth						
In debt						
\$50K–\$199,999						
\$200K–\$499,999						
\$500K–\$999,999						
\$1 million+						
Intercept	0.61***	0.031	0.62***	0.030	0.65***	0.031
Persons	10,197		10,197		10,197	
Person-Years	56,262		56,262		56,262	

Note: Data are adjusted for all covariates, including proxy, ever died, and item missingness.

† *p* < .10; * *p* < .05; ** *p* < .01; *** *p* < .001

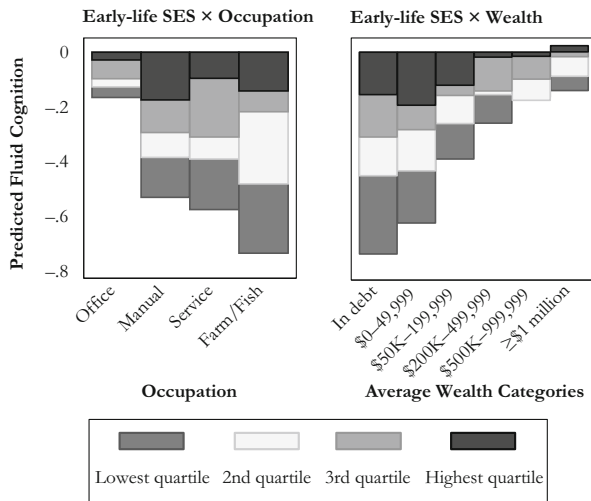


Fig. 3 Cumulative inequality model. Predicted fluid cognition (at age 80) from interactions between early-life SES quartiles, occupation, and wealth, adjusted for covariates

July 1930–1934; $b = -0.29, p < .001$). The cohort pattern and estimates for the other predictors are consistent. I also estimate models using single birth-year cohorts. Comparing neighboring birth-year cohorts minimizes problems with selective mortality and approaches a more fine-grained analysis of exposure. There is a significant dip in fluid cognition for those who were born in the year following the stock market crash relative to those born the previous or following year. Compared with the reference group born in 1918, birth year coefficients are $b = -0.245$ for 1929, $b = -0.290$ for 1930, and $b = -0.264$ for 1931 ($p < .001$). As additional sensitivity analyses for mortality selection, I conduct simulations (Online Resource 1, Figs. S2 and S3).

If cognitively impaired elders are more likely to misreport their early-life SES negatively, low early-life SES would inaccurately appear predictive of lower fluid cognition. This direction of misreporting may also bias the cohort estimates toward

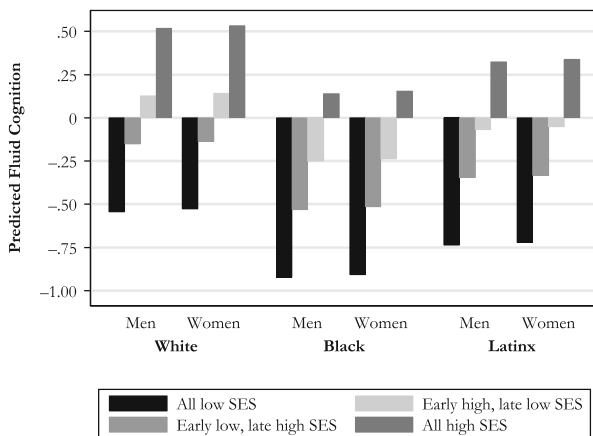


Fig. 4 Cumulative inequality model. Predicted fluid cognition (at age 75) for black, Latinx, and white men and women by life-course SES

the null because earlier cohorts enter at older ages and average worse fluid cognition at first TICS. At their first eligible TICS test,¹⁷ 1,439 (11.7 %) and 517 (4.2 %) respondents (of $n = 12,278$) fall 1 or 2 standard deviations, respectively, below the fluid cognition mean. As a robustness check, I reestimate all the models but exclude individuals whose first test suggests impairment.¹⁸ Although the cohort and sex coefficients marginally increase, there is a modest decline in coefficients for some of the other factors, including race/ethnicity, family SES, and occupation. Nevertheless, estimates are consistent, and sex is the only variable that changes in significance (Online Resource 1, Tables S1 and S2).

Cardiovascular diseases are associated with early-life hardship (cf. Scholte et al. 2012); therefore, I test the subhypotheses that exposure to the Depression increases BMI, as well as risk of stroke, high blood pressure, heart disease, and diabetes. I find mixed results for these models, but overall cardiovascular health does not appear to be the mechanism connecting early-life exposure with fluid cognition (Online Resource 1, Table S3).

Finally, I use historical Census Bureau data to examine early-life contextual factors. I include childhood region's average unemployment rate during the early 1930s to account for regional variation in severity of the Depression. Unemployment ranged from 20 % to 28 %. Fluid cognition is statistically significantly associated with, but not patterned by, regional unemployment. This suggests the variable is capturing other regional dissimilarities. I model fluid cognition as a function of total birth rate, infant mortality rate, and child mortality rate to assess the possibility of a culling effect (Myrskylä 2010). I find no indication that cohort estimates are downwardly biased (i.e., more conservative) because of selection in early-life birth and death rates (Online Resource 1, Fig. S4).

Discussion

Framed by life-course theory, I test whether social factors affect later-life fluid cognition through processes reflective of critical period, social trajectory, and/or cumulative inequality models (CPM, STM, and CIM, respectively). I find individuals exposed to the Great Depression at vulnerable ages have lower fluid cognition than those over age 10 at the stock market crash, net of age, life course factors, and controls (Table 2)—evidence for the CPM. However, exposure to the Depression is not significantly associated with rate of cognitive decline.

I find little evidence for the STM. Net of controls and compared with the most insulated cohort (born 1909–1918), the two most vulnerable cohorts (age 2 and under at the stock market crash or born during the Great Depression) have higher education (decreases risk) and occupational attainment (decreases risk), but the Great Depression cohort is more likely to consume three or more alcoholic drinks per day (increases risk) (Table 3). The CPM shows that these behavioral factors attenuate some of the other risk factors but do not significantly affect the cohort coefficients (Table 2).

¹⁷ Measured either at Wave 3 or at entry into the survey if entry is thereafter.

¹⁸ This sensitivity check is conservative because I exclude people who were impaired at Wave 3 even if they answered the early-life SES questions at Wave 1.

I find evidence for the CIM. Multiple aspects of poverty over the life course—early-life exposure to the Depression, poor childhood health, lower parental and respondent education, lower occupational attainment, and lower adulthood wealth—accumulate to predict multiplicatively lower fluid cognition (Fig. 3). People of color have worse fluid cognition than whites across all SES groups (Fig. 4), and blacks' fluid cognition declines faster. In short, all evidence points toward the accumulated negative effects of a disadvantaged life, including exposure to macroeconomic shock during ages critical to neurocognitive development.

Limitations

This study has limitations. Because TICS is a brief telephone-administered cognitive assessment, it is not as effective at evaluating cognitive function as clinically administered tests. Although TICS has been validated as a good indicator for pathological cognitive decline with high sensitivity and specificity (Crimmins et al. 2011; Crooks et al. 2005; Fong et al. 2009), this study cannot make strong claims that Alzheimer's disease or other pathological cognitive decline caused low fluid cognition scores.¹⁹

An additional measurement limitation is that there are plausibly practice effects for the TICS. Respondents may perform better over time because they are familiar with the questions. This would downwardly bias estimates, making cognitive decline seem less steep. Vivot et al. (2016) found that associations between risk factors and cognitive decline are not significantly affected by practice effects.

Early-life factors are self-reported retrospectively, opening them to recall bias. However, respondents answer early-life questions at their first interview, when they are least likely to be suffering from cognitive impairment or comorbidities. Also, Alzheimer's disease and other age-related cognitive impairment affect short-term memory and problem-solving skills much earlier than long-term memory (Buckner 2004). A respondent will thus be unable to answer the current questions well before their early-childhood memories are compromised (Buckner 2004; Greene and Hodges 1996), in which case the interviewer requests a proxy, and the respondent does not complete the TICS, excluding them from these analyses.

Nevertheless, I use two strategies to address possible recall bias. First, instead of operationalizing early-life SES with a single question (e.g., parents' education), I include multiple domains of early life. This should be less sensitive to misreporting. The CPM encompasses six separate measures of early-life SES (see Table 2 and Fig. 4), and the CIM uses a composite measure of early-life SES (see Table 4 and Fig. 3). Second, in robustness checks, I exclude elders who are either 1 or 2 standard deviations below the fluid cognition mean at first TICS test. Although there is modest fluctuation in some coefficients (see the sensitivity analysis in Online Resource 1, and Tables S1 and S2), results are consistent. Measurement error in retrospective reports cannot be eliminated. In fact, missingness on covariates is associated with SES ($b = -0.12$, $p < .001$), so estimates for SES could be downwardly biased.

¹⁹ The HRS's subset Aging, Demographics, and Memory Study (ADAMS) includes a larger battery of cognitive tests but has smaller samples and primarily recent cohorts (cf. Boardman et al. 2012; Gomar et al. 2011; Tanzi 2012).

To manage selection into the sample and selectivity, I include indicators for proxy and mortality attrition, restrict analysis to those born 1909 to 1934, examine cohorts within the same age range, and use counterfactual simulations. Even so, bias may remain. If healthier individuals make up a greater share of earlier cohorts because they have survived to older ages, cohort estimates will be upwardly biased. On the other hand, if those most negatively affected by the Depression were selected out of the study by premature mortality, the bias would be toward the null, and estimates linking fluid cognition with exposure to the Depression or poverty would be conservative.

Conclusion

The entire life course is essential for understanding health disparities (Corna 2013; Crosnoe and Elder 2004; Kaplan et al. 2001; Lynch and Smith 2005; Turrell et al. 2002; Wadsworth 1997). I argue that the emphasis on education to predict later-life cognitive function is overstated and that life-course SES, more broadly, impacts cognitive health. Indeed, in this case, a more complex picture emerges.

I study three theoretical models for this process: the CPM, STM, and CIM. I find little evidence for the STM that behavioral mechanisms connect early-life exposure to the Depression with later-life fluid cognition. I do find evidence for the CPM in that no covariate significantly attenuates the association between cohort-exposure to the Depression and fluid cognition. However, evidence for the CIM demonstrates that life-course disadvantages accumulate to affect later-life fluid cognition and that modifiable social factors may promote cognitive resilience or ameliorate low early-life SES.

This research makes several contributions. The large, nationally representative HRS sample, born over three decades, enables investigation of individual-level and macro-economic factors. Using the Great Depression as a quasi-natural experiment adds to our understanding by targeting cohorts who were exposed to macroeconomic shock at different ages of neurocognitive development. This approach enables analysis of how early-life exposure to exogenous shock and the long-term accumulation of disadvantage are associated with cognitive health. Finally, the interaction effects among early-life SES, occupational attainment, and wealth (Fig. 3) highlight a key implication—the importance of examining interaction effects in life-course research.

This study signals several areas for future research. When cohorts born after the Depression are old enough to assess later-life cognitive decline, research should include these unexposed cohorts. The Depression did not have a uniform impact on the country; some regions and families therein experienced greater hardship. However, in this analysis, regional unemployment and interactions of cohort with early-life SES and region yield little explanatory value. Future research could add validity by incorporating life histories that detail exposure to early-life hardship.

Racial/ethnic disparities in fluid cognition are significant despite controlling for life-course SES and chronic illnesses, and blacks have a faster rate of decline. The latter may be evidence of *weathering*, the hypothesis that people of color age faster because of the detrimental health effects of stress exposure (cf. Geronimus et al. 2001, 2015). This possibility should be investigated.

Future research that includes more precise measures of the role of partnership and other social interactions could produce valuable insights. For women, widowhood and

separation/divorce/absent spouse are associated with higher fluid cognition than partnership. Women and widows are more likely to socialize externally (Waite and Das 2010), which may be more protective than familial interactions (Glei et al. 2005), but it is not clear why. Are couples more likely to compensate for a partner's declining fluid cognition, promoting deficiencies as elders lose capacities they do not use (Vemuri et al. 2012)? The positive relationship between higher BMI and fluid cognition could reflect selective mortality, a confounding variable, or an insulative effect against cognitive decline that warrants further examination (cf. Doehner et al. 2015 for a similar paradox in cardiovascular health). Future research should also investigate why occupational attainment and wealth partially compensate for early-life poverty.

Already in 2015, 47.5 million people worldwide had a dementia diagnosis, which is predicted to triple to more than 135 million by 2050 (World Health Organization 2016). This study adds robust empirical support for interventions that mitigate macroeconomic shock, especially for pregnant people and young children. In addition to the more widely studied education, policymakers ought to consider malnutrition, allostatic load, and other SES-linked factors as vital intervention points. The apparent benefits of later-life socializing and exercise demonstrate the importance of supporting elders and increasing their social integration. In conclusion, population aging, dire predictions of dementia prevalence, and the lack of a cure for Alzheimer's disease demand a closer analysis of modifiable social factors.

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